Byron Hyde M.D.
The Nightingale Research Foundation
121 Iona Street,
Ottawa, Canada,
K11Y 3M1

Tel. (613) 729-8995
Fax. (613) 729-0148
Email: bhyde@nightingale.ca Secretary (613) 728-5490 (home)

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Michael A. Roberts,
Graydon Head & Ritchey LLP
Attorneys at Law,
1900 Fifth Third Center
511 Walnut Street
P.O. Box 6464
Cincinnati, Ohio,
USA, 45201 - 6464

Tel. (513) 621-6464, direct line: 629-2799
Fax. (513) 651-3936?? Email mroberts@graydon.com

Re: Mr. Eric Jeffries

Dear Mr Roberts,

I am sorry that I was not able to start the report that you requested in the week of my departure to speak in the UK. The week was largely taken up with last minute patients and their medical matters and preparing the slides for my talks here.

I have taken your letter with me with the intention of replying to it but I have not brought the file of Mr Jeffries since I simply had not time to have it copied and did not wish the chance of losing it. So this letter will be from memory and I will correct it on my return after the 15th of November if need be

Let me begin by stating that I have not completed my investigations of Mr. Jeffries due to the obvious hiatus caused by my discovering his thyroid malignancy and the importance of first attending to that matter. As you know he has had this thyroid malignancy removed and is now undergoing therapy to abort any metastatic malignancies that might have resulted from this problem

Second, although your request is most reasonable in asking for a report it is not my nature to do so until I have completed my investigation as far as it reasonable possible. The reason is simple. Many of these cases that I have seen are very complex and it is a little bit like opening up one of

those Russian Babushka dolls in which we may discover a new doll or a new pathological condition inside each doll that is opened. In the case of Eric Jeffries I have opened up the first doll and found a generalized and severely abnormal immune dysfunction. When I opened that doll I found what appears to be an inflammatory reaction to the arteries of the brain and another doll with an inflammatory reaction to the thyroid. When I opened up the inflammatory thyroid doll I found a malignancy. As you can see I have just begun this somewhat frightening game of discovery. For these reasons you have to bear with me in that I cannot tell you yet how much more that I, with the help of my associates, will find. Until I am sure there are not more pieces to this puzzle it is difficult to be certain how they will fit together.

Having said that there are certain obvious opinions that I can give and shall do so.

You wished a response for the following questions. "I would like you to set forth in summary fashion your: (1) Work to date: (a) I have had approximately 20 telephone conversations with this Mr. Jeffries prior to, during and after I have examined him. I have also reviewed the statements of many of the physicians who have examined Mr. Jeffries as well as many of the tests and reports that these physicians originated. I then ordered certain consultations with physicians who I believed were appropriate to his case. I also took a careful history and physical examination and made observations of the patient over a period of a good portion of approximately two days. This is my normal approach with each of these new patients who come outside of the normal easy daily driving distance of my office. (b) I then ordered certain tests and examinations that I believed to be appropriate in this case. These tests involved in depth examination of the patient's immune system, his cardiovascular system, his neurovascular system, certain aspects of his endocrine system and his blood, urine, stools and chemistry.

(2) Observations to date: (a) History: From his various prior medical histories and that of the patient it was obvious that Mr. Jeffries had an acute illness involving a significant upper respiratory track illness but no history of any serious illness or chronic ongoing disability until he received a combination Hepatitis A and B immunization during the height of this acute infectious disease. May I point out that certain immunization protocols that come with the immunization kit specifically contra-indicate the immunization during an obvious infectious disease. I have requested that Mr. Jeffries obtain a copy of the appropriate combined A and B protocol and instruction paper that comes with the vial of immunization suspension. I have not received this paper although I have received a verbal account of the contents. I would prefer not to comment further on this matter until I see the actual immunization instruction paper. (b) Adverse Immunization Reaction: In the next few days following the double Hepatitis A & B immunization Mr. Jeffries was not clinically worse. Then in a time period consistent with an adverse immune type reaction he developed a series of problems that I have observed in a large number of patients following Recombinant Hepatitis B immunization during the past 14 years. These include the development and persistence of memory dysfunction, speech dysfunction, various cognitive dysfunctions, loss of anticipated pre-illness intelligence levels, rapid fatigability, SPECT and PET and Neuropsychological abnormalities. This is a partial list of his intellectual and cognitive changes.

(c) NeuroSPECT: Changes noted include a vasculitis-like pattern of the CNS that I have seen only in those patients with worse case scenarios. This probably relates to a low-grade inflammatory reaction of the CNS arterial bed. There were also significant PET scan views of his brain physiology however I do not have the report with me as previously noted and will include these on my full report. (d) Endocrine Dysfunction: I found in the initial physical examination what appeared to be a minor thyroid irregularity with possible nodules but in the first series of the immune testing that although the normal thyroid tests were either normal or mildly abnormal, the autoimmune antibodies were of major proportions suggesting that the body was attempting to reject or attack

it's own thyroid gland. Any amount of antithyroid antibody is abnormal but this degree of anti thyroid activity is grossly abnormal. To find abnormalities of thyroid activity is relatively common in patients with post - hepatitis B immunization disease. I then organized a thyroid uptake scan that suggested Graves's disease and abnormal activity. The patient clinically resembled Hashimoto's Thyrotoxicosis and not Graves's disease. A needle biopsy was performed and the diagnosis came back as follicular cell carcinoma. When the thyroid gland was removed the definitive diagnosis was given as areas of follicular cell carcinoma and Hashimoto's Thyrotoxicosis. Conclusions: It is too early to come to any other conclusions other than the obvious. It is my opinion that: (1) It is medically improper to give an immunization and particularly a multiple immunization to any patient with an active infectious disease. This is particularly so if one does not first ascertain the nature of this primary infection. Since in my experience many of the severe and permanent adverse immune reactions resulting from Recombinant Hepatitis B immunization have occurred when the patient already had a primary acute, subacute or chronic infection at the time of immunization.

(2) I have seen significant SPECT changes in the Central Nervous System (CNS) that appear to be a vasculitis pattern or inflammatory pattern following immunization with Hepatitis B; I have seen many patients with pathological thyroid disease as well in a large number of post Hepatitis B immunization patients. There are an increasing number of publications in medical journals over the past 10 years associating Hepatitis B immunization with endocrine and CNS disease. It is my opinion that there is a direct relationship between the immunization of Eric Jeffries with the Hepatitis A and B immunization combination while he was ill with an infectious illness and his subsequent and chronic CNS and endocrine injury.

(3) It is my opinion that Eric Jeffries suffers and has suffered from an autoimmune disease caused by the immunization of Hepatitis A and B. Since I have to date seen no adverse effects of Hepatitis A immunization it is most likely that the Hepatitis B component was the cause. I must add that Hepatitis A immunization is a relatively new addition and since it is being given at times with Hepatitis B immunization it may be difficult to come to any final conclusions at this early date on Hepatitis A.

(4) It is my opinion that Eric Jeffries has been disabled increasingly after the combined Hepatitis A and B immunization and that from the time he was obliged to cease all work and until the present time he has been totally and completely disabled from being able to work at his former occupation.

(5) Due to his present treatment for cancer and the side effects from this treatment he now has an increased number of medical problems. Until this treatment has settled down and until his thyroid has been stabilized I will be unable to continue with my investigation. It is my opinion that Eric Jeffries is disabled for the foreseeable future and it is impossible for me to come to any additional conclusions until he has got through the next year of treatment and investigations.

I do apologize for this delayed report but I am on the road and this is a borrowed computer. You can reply to this address but after Monday night I will be in Newcastle upon Tyne and will return to this address briefly prior to leaving for Paris to speak with some physicians there who are working on Hepatitis B immunization.

I will not be able to supply my CV until I return.

Yours sincerely,

Byron Hyde M.D.

Byron Hyde, M.D.
The Nightingale Research Foundation
121 Iona Street, Ottawa, Canada K1Y 3M1
Tel: (613) 729-8995 • Fax: (613) 729-0148 • E-mail: bhyde@nightingale.ca

June 30, 2001

Eric Jeffries
2338 Bedford Ave.
Cincinnati, Ohio, 45208
USA

Dear Mr. Jeffries:

This is my up-to-date report on my analysis and diagnosis of your illness. I understand that this will be shared with others and, therefore. I have attached my Curriculum Vitae.

I. Summary of Diagnosis and Conclusions

Until 1997, by all accounts, Eric Jeffries was a healthy and successful banker. In 1997, during a time that Mr. Jeffries complained of a sore throat, he received an immunization for Hepatitis A and B. Within a week, he became increasingly ill and since that time, he experienced a downward spiral of his health, which led to his permanent departure from work in September 1998. Since June of 1997, Mr. Jeffries has seen many physicians and undergone many tests. My treatment and observations of Mr. Jeffries began in June 2000 and continues through today. My examination. observation. and review of Mr. Jeffries lead me to make the following conclusions:

(1) Mr. Jeffries has an evolving, ongoing immune mediated injury. The areas most injured appear to be to his Central Nervous System, specifically his brain but also his red blood cells and his thyroid gland:

(2) Eric Jeffries has been and remains seriously ill. His chronic illnesses started shortly after his immunization and gradually became increasingly disabling. He remains significantly disabled with major and minor variations. He improves after rest but these improvements are ephemeral. During the past three years or more he has experienced no periods of sustained recovery. It is impossible to say at this time whether Mr. Jeffries will ever recover. It is important to watch him carefully since his situation is unstable and I cannot rule out the development of another malignancy.

(3) It is my opinion that Mr. Jeffries' illness totally disables him from returning to his previous employment or intellectual work of any kind. Furthermore, it is my opinion that this total disability has persisted from at least the time Mr. Jeffries ceased employment in September 1998, and will continue into the foreseeable future.

II. Patient's History

Mr. Jeffries was born in Missouri on the 15th of May 1961, and was a strong and healthy baby. At the age of four he contracted Rocky Mountain Spotted-Fever. Eric went through primary school without any medical problems. In grade 9 he was in a car accident resulting in a broken scapula and dislocated shoulder. He was very active and gifted student athlete in sports, including football, wrestling and track and field. He had several minor sports related injuries and an injury to his neck while playing football for his university. He has no other history of significant illness or injury. He has never taken any addictive medications or had any history of psychiatric disease. He had never had any surgeries at the time of his initial examination. His records indicate that he began experiencing epididyimitis circa 1993, and he also recalls this as being the approximate time in which he began developing mouth ulcers. In March 1995 he notes that while at his physician's office he reported feeling unwell with hot flashes and with slight disorientation and shooting pain up the back of his neck. This complaint did not persist and is consistent with a minor viral infection. He also notes that shortly prior to falling ill, his physician had remarked on an increase in his serum bilirubin levels but that this was apparently considered to be of no medical importance at the time. He has always been both strong and healthy until the present illness.

III. Genetic Factors

Both of Mr. Jeffries' parents are living. In 2000 his 56-year old mother was healthy except for a remote history of uterine and back surgery. Both she and her brother (Eric's maternal uncle) have a problem with chronic furunculosis and it is possible that this may be due to a minor immune disorder. Mr. Jeffries' father (age 59) is now blind and deaf as the result of degenerative genetic disease, which affects his CNS. Both he and his brother (Eric's uncle have this recessive and X-linked genetic disease affecting the CNS. It is termed Usher's Syndrome and causes progressive deafness and loss of vision. A progressive loss of intellectual abilities occurs in 25% of the patients with this genetic condition. Eric's immediate family also have a second genetically inherited disease, Retinitis Pigmentosa. This is a CNS disease, usually autosomal recessive and affecting the photoreceptor layer of the retina. It usually begins slowly in childhood. There are many forms and associations of this genetic disease. One form causes spinocerebella and cerebellar ataxia. Eric's great aunt (paternal) also suffers from Ushers Syndrome. There is no evidence that Eric has either disease process, although Eric does have some degree of ataxia. However there may be a susceptibility to autoimmune illness in this family.

Mr. Jeffries has four siblings, one full sister and three half brothers. His full sister, Kathy (40 years old), is a housewife who has an autoimmune disorder diagnosed as probable Lupus with a positive ANA. His three half brothers are thought to be healthy with no known illnesses.

Eric's maternal grandmother, age 83, had polio as a young woman. She is in relatively poor health with chronic lymphocytic leukemia and a non-specified arthritis. The maternal grandfather died at 92 with a broken hip and no illnesses. However, the maternal grandfather's siblings had Alzheimer's disease. Eric's paternal grandmother is 82 and healthy. The paternal grandfather died at 85 with heart failure and prostate carcinoma.

In other words, although Eric has none of the family illnesses noted when examined by me, his genetic history is not fortuitous. Also, we have not yet done tissue typing and chromosome analysis of Eric. This is planned for his next visit to Ottawa. Alzheimer's Disease does have a genetic component with increased incidence in the family of Alzheimer's patients. There is no satisfactory test for early Alzheimer's.

IV. The Onset and Progression of the Illness

Prior to his 1997 immunization, Mr. Jeffries had an increasingly sore throat for two or three weeks. Mr. Jeffries saw his physician in order to have this sore throat treated. His physician in addition to treating the sore throat also administered a combined Hepatitis A & B immunization. Immediately after receiving the vaccine, Eric boarded a plane for a business meeting in Dallas, Texas but had to cancel the meeting upon arrival due to an onset of acute illness. Approximately one week later, Mr. Jeffries was overcome by a severe headache with strange body pains, night sweats, lethargy, difficulty in concentration and face pain.

These symptoms persisted and Mr. Jeffries' physician contacted SmithKline Beecham, the maker of the immunization given, to inquire about adverse reactions. The vaccine maker acknowledged other reports of similar symptoms and suggested that it was serum sickness, which should not persist. I believe the physician was also told not to repeat the immunization as Mr. Jeffries might react in an untoward way to further immunizations of this nature.

Serum Sickness: A hypersensitivity reaction to the administration of a foreign serum or serum proteins characterized by fever, urticaria, arthralgia, edema and lymphadenopathy. It is caused by the formation of circulating antigen-antibody complexes that are deposited in tissue and trigger tissue injury mediated by complement and polymorhonuclear leukocytes. Also Serum Sickness-Like Syndrome from antibiotics and other drugs. **Dorland's Medical Dictionary.**

Causes: Drugs and various immunizations. Symptoms: Above plus myalgias, extremity weakness, myocarditis: Possible Complications: Vasculitis, Neuropathy, Death. Treatment: Corticosteroids if peripheral neuritis if antihistamines do not provide relief or if myocarditis occurs. **Griffith's Clinical Consult**

After a period of brief recovery, these symptoms struck again with muscle pains and numbness in his arms and legs, particularly on the right side. During the first year of the illness, Mr. Jeffries had recurrent bouts of this type of symptom complex, but after each bout he appeared to stabilize at a new low level of physical and intellectual ability.

The symptoms and pattern of the illness Mr. Jeffries experienced were consistent with both autoimmune and CNS vasculitis disease. He sought the medical expertise of several physicians in an effort to recover. However, the illness continued to progress in an intermittent, repetitive increasingly disabling fashion until the point in September 1998 at which Mr. Jeffries could no longer work.

Mr. Jeffries continued seeing physicians in an effort to regain his deteriorating physical and cognitive health without improvement. He then also sought help through alternative medical practices including acupuncture, herb therapy, and mystic healing. Alternative medicine was also of little use.

While the physicians that examined Mr. Jeffries recognized he was suffering from an organic illness, it proved to be difficult to precisely define and treat. During this period many diseases were ruled out, although numerous objective and clinical abnormalities continued to surface.

V. Physical Examination

I have examined Mr. Jeffries on two separate occasions, June and December 2000. During the first examination I discovered a nodule on the right lobe of his thyroid, which, through subsequent investigation, was found to be cancerous. The thyroid was also involved in an autoimmune inflammatory breakdown diagnosed as Hashimoto's Thyroiditis. The carcinoma, thyroid, and surrounding lymph nodes were removed shortly thereafter. The pathological report confirmed both diagnosis of malignancy and Hashimoto's Thyroiditis.

Below is a summary of the findings from the most recent examination on 6th December 2000:

Blood Pressure: Dysautonomia

	Supine	Seated	Standing
Brachial Pressure	120/80	115/82	105/90
Pulse Pressure	40	33	15

Although his brachial pressures are normal to subnormal his pulse pressures are not normal. His falling pulse pressures may be indicative of dysautonomia. This can be suggestive of an injury to the brain in the area of the subcortex. The significance of this particular diagnosis is that a probable CNS injury has occurred that makes it difficult for Mr. Jeffries to maintain normal blood pressure and normal circulation to his brain.

General Appearance: he has Parkinsonian like faces; he is not depressed; he has no lupus rash; he is kempt. The patient walks with a wide leg stance and is in obvious discomfort. At times he limps. He cannot keep up to me even when I walk slowly in the hospital. He appears clumsy in his walking. He has an abnormal speech pattern with almost scanning speech. He does not talk normally. He gets lost in mid-sentence and frequently cannot remember what he was saying to me.

ENT: normal dentition and pharynx and auditory canals. No postnasal drip but very dry pharynx. He has a tremor of the tongue when extended. The scar from the thyroidectomy is still rigid.

Lymph Glands: absent or not palpable cervical, axillary and inguinal lymph glands.

Cardiac examination: normal valves, rhythm and heart sound and regular pulses. Falling pulse pressures with standing.

Lungs: normal.

Abdomen: normal, no tenderness and no visceromegally.

Rectal/Scrotal: very dry anal sphincter with small prostate and with no haemorrhoids. Tender testes.

Extremities: hands normal with no suggestions of finger blanching or discolouration.

Spine: he has a rigid spine with no evidence of surgery or scoliosis. There is a marked cervical lordosis with a pronounced dowager's hump.

Fibromyalgia and muscle strength: he has no generalized musculoskeletal aching apparent on examination and 0/18 tender points. He is obviously stiff and has decreased lower right leg strength. His muscle pain is cyclical but he does not appear to have any fibromyalgia type illness.

Eyes: there is no argyle Robertson pupil. He has bounding anisocorial pupils (unequal diameters) and contrary papillary reaction in both eyes. There is no ptosis or nystagmus. No obvious Sicca syndrome. Sclera are normal.

Neurological Exam: there is a minor bilateral tremor. This exists also in his tongue. He has right leg cogwheel type leg risings immediately but none in the left leg until the 7th leg raising. He has problems of clumsiness but no Romberg sign. Both his left and right hand misses by two inches on finger-finger exam. He has a right plantar equivocal reflex the left is normal. His right leg went into spasm when examined.

Speech: He has inappropriate words, repeated words and phrases, and slurring of speech.

Results: He had problems of balance and mobility but no Romberg. He was clumsy. He had a decrease flexor strength and a bilateral mild tremor. He has some abnormal reflexes but mainly hyporeflexic that may be consistent with his thyroid or CNS disease. He had a contrary pupillar reflex and other minor eye reflex abnormalities that might be consistent with the abnormal SPECT findings. He had right leg cramping on examination. The flexors of his right arm were less powerful than the left. He had cogwheel movement of his legs right worse than left. He has problems with past pointing. He had a falling pulse pressure on standing. He has a wide legged Parkinsonian like walk and manner but no obvious Parkinson's disease. He has thick facial skin and moon shaped face that might be due to the thyroid disease or other endocrinological abnormality.

None of the above findings are hard clinical signs for a specific CNS injury or a specific CNS disease: these findings are consistent with a widespread diverse CNS injury or illness affecting Eric's brain and possibly spinal cord. These findings are in part also consistent with endocrinal damage. There is solid test evidence that such pathological changes have occurred.

This lack of any major physical signs is quite typical of M.E./CFS like disease and merely rules out space occupying disease or gross demyelination diseases. He has several curious minor neurological abnormalities. He has also has Parkinsonian features but not Parkinson's disease.

Review of Insurance Film: I reviewed a film taken over several days during the period of December 6th to January 8th 1999 –2000, by agents of his insurance company or companies. This film is quite revealing in that at no time does he appear normal. He is obviously in pain in several of these episodes, he walks with a stiff limbed almost Frankenstein walk. Often he limps suggesting joint or back pain. His head is bowed over, his upper thoracic spine is humped or hunch backed and it is obvious that walking is difficult for him. His time reactions are always slow and often clumsy. When another adult or his children accompany him, the others get out of the car at a regular speed and Mr. Jeffries is left far behind. At times he walks with a wide stanced walk. He looks and acts like a typical Parkinsonian brain dysfunction with slowing and irregularity of all motor functions. He has difficulty both getting in and out of his car and on the January 8th segment when he attempts to close the car-door, he clearly misses the handle completely. At other times in the garage scenes he simply stands in a doorway like a statue or a

patient with Alzheimer's disease, without moving for long periods of time as though he was lost. This patient has a definite motor problem and a definite balance problem and probably a subcortical injury. He has a movement disorder but although this may be an early Parkinson's disease it is more likely a result of the subcortical and cortical vasculitis like pathology seen on the SPECT scans. From the abnormal SPECT Scans I would suggest he has a significant CNS injury-giving rise to this non- Parkinson movement disorder.

I have not had access to all of the consultations attended by Mr. Jeffries. I append some of the relevant tests and consultation that I do have.

V. Tests Reviewed.

I do not believe that I have all of the tests performed on Mr. Jeffries but the tests I did review are more than sufficient to demonstrate major illnesses. I will supplement this report upon review of any records of significance, which may be directed to me.

A. July 1997: Hepatic panel:

Type of Test	Test Result	
SGOT 36 (primarily heart enzyme)	Abnormal	(normal 11-35)
SGPT 59 (primarily liver enzyme)	Abnormal	(normal 7-46)
Bilirubin total unconjugated 1.7	Abnormal	(normal 0.2-1.2mg/DL)
Direct conjugated bilirubin 0.3	Normal	(normal 0.0-0.3
CK (Creatine kinase) 360 U/L	Abnormal	(normal 48-251 U/L)
Reticulocyte count 1.7	Normal	(normal 0-15)

(These abnormal tests are suggestive of a liver dysfunction, disease or inflammation at the time. This could be viral, immunological, inflammatory, infectious, or secondary to immunization disease or a combination of any of the above.)

B. 24th June 1998: Health Alliance: Dr. Dunn

The total unconjugated bilirubin was 2.0	ABNORMAL	(normal 0.2-1.2)
Direct protein conjugated bilirubin was 0.3	NORMAL	(normal 0.0-0.3)

Discussion: Increased bilirubin can result from the breakdown of haemoglobin in the red blood cells and is a by-product of red blood cell destruction. Thus a rise in serum bilirubin will occur if there is an excessive destruction of RBCs or if the liver is unable to excrete the normal amounts of bilirubin produced. It can occur in other diseases of the liver as well. Mr. Jeffries has indication of a pathological red blood cell breakdown, possibly secondary to an autoimmune injury to the red blood cells.

Unconjugated bilirubin is associated with red blood cell destruction. The bilirubin test taken in the first month after immunization was abnormal and Mr. Jeffries' liver enzymes were elevated. We can say with some assurance that by July 1997, one month after the immunization and on June 1998, a year later, there was evidence of abnormal liver involvement or disease and/or increased red blood destruction and increased abnormal activity in the liver due to this.

Because more specific tests do not appear to have been performed or recorded at that time it is not possible to say whether the liver was damaged at the times mentioned above or if it was increased red blood cell destruction or both. Since I have been able to determine the existence of increased reticulocyte formation on all tests done in Ottawa in 2000 and earlier but not immediately after he fell ill, unless there is some other explanation to the contrary, it is logical to assume that an autoimmune or other destruction of the red blood cells may have been going on increasingly since Mr. Jeffries was first tested in the first month following the Hepatitis A & B immunization in June 1997 and that this became apparent only later. It would be interesting to review serial tests of liver function and bilirubins and cold agglutinins if they were consistently performed. This would suggest the real possibility of an immunization associated autoimmune injury starting at one month after the immunization but slowly progressing in degree.

CK (Creatine Kinase): The primary source of this enzyme is brain and both cardiac and smooth muscle. It is elevated in virtually any ischemic injury or muscle inflammation. Injury to the brain releases CK. This enzyme is increased after myocardial infarct and remains elevated for no more than 36 hours after an infarct. It is thus highly unlikely that this patient's CK elevation was due to an infarct, particularly since the SGOT was virtually normal. CK also elevates due to hypothyroidism, central nervous system trauma, myositis and due to Reyes syndrome. However no SGOT/SGPT appears to have been performed immediately at the time his illness started, and no serial tests appear to have been performed, therefore it is impossible to be certain whether a minor heart injury occurred that has since healed. This is unlikely to have occurred and probably we are seeing the results simply of the liver and CNS injury. Myositis is ruled out due to the normal sedimentation findings. However many of these tests were performed inconsistently so it is difficult to follow. This leaves Reye's-like syndrome, CNS injury and hypothyroidism as possible causes.

Definition: Reye's syndrome: Usually seen in childhood, with elevated transaminase tests and changes in the liver; an encephalopathic condition may follow with disturbances of consciousness. Recovery often occurs leaving sequellae.

Blood Pressure recordings:

Position	Blood Pressure R. arm	Pulse Pressure mm. Hg
Lying	120/80	40
Sitting	115/82	33
Standing	105/90	15

Discussion: Although the arterial blood pressures are normal the pulse pressure falls when Mr. Jeffries changes from a supine to sitting to a standing position. This is an abnormal finding. It may suggest an abnormal autonomic status in which the subcortical brain has lost its ability to create a normal pressure response in the extremities.

C. Blood Volumes: Nuclear Medicine: Hotel Dieu Hospital, Montreal: Circulating Blood Volumes: 13th June 2000.

Circulating Volume	Normal Finding adjusted for surface measurement	Patient finding	Percentage of Normal	Estimated Standing Circulating Volumes
Total Blood	7.11 Litres	5.48 Litres	78%	58-68%
Red Blood Cell.	2.84 Litres	2.40 Litres	85%	65-75%
Plasma	4.26 Litres	4.27 Litres	72%	52-62%

Discussion: These tests were all taken with the patient lying down. It is our experience that when sitting, the figures decrease by a factor of 5-10% and when standing they decrease by a further 5-10%. What this means is a standing circulating Red Blood Cell volume would be anticipated to be in a range of 65-75% of normal. Similarly the plasma volume would reduce to 62-52% of normal when standing. This pathological decrease in circulating red blood cell and plasma would have a profound effect on the patient's ability to deliver red blood factors such as oxygen and plasma factors such as insulin and growth hormone to name two, for adequate cell metabolism and specifically normal brain metabolism. This could certainly contribute in part to Mr. Jeffries decreased intellectual and energy abilities. It is unknown why his circulating red blood and plasma volumes should be so low. It suggests pooling of his blood in the legs or the abdomen. This is pathological and it may suggest that his modest autonomic findings are being underreported. For instance when we did the tests the patient was not belted in and if he moved this would have negated the results to some degree. I assume that he has greater autonomic dysfunction than our tests reveal. He also has a low-grade red blood cell destructive immune injury. It is not known if this is related to the above findings.

The above findings are pathological and suggestive of subcortical brain injury.

D. Immune Tests:

1. Status: 1st measurement: June 19, 2000: CHEO: Children's Hospital of Eastern Ontario.

Immune Cell Type	Normal Range	Patient Test Result	Normal Abnormal
CD3 (Total T cells)	1100-1700	**987**	Abnormal
CD4 (Helper T cells)	700-1100	**629**	Abnormal
CD8 (Supp. T cells)	500 - 900	**358**	Abnormal
CD19 (Pan-B cells)	200-400	**172**	Abnormal
CD4/CD8 ratio	1.0-1.5	**1.8**	Abnormal
CD16+56 (NK cells)	200-400	215	Normal (*)
Absolute Lymph cells	1.6-2.4	**1.43**	Abnormal

(*) activity unknown

2. 2nd measurement:

December 21, 2000: CHEO: Children's Hospital of Eastern Ontario.

Immune Cell Type	Normal Range	Patient Test Result	Normal / Abnormal
CD3 (Total T cells)	1100-1700	**893**	Abnormal
CD4 (Helper T cells)	700-1100	**562**	Abnormal
CD8 (Supp. T cells)	500 - 900	**331**	Abnormal
CD19 (Pan-B cells)	200-400	**130**	Abnormal
CD4/CD8 ratio	1.0-1.5	**1.7**	Abnormal
CD16+56 (NK cells)	200-400	**360**	Normal (*)
Absolute Lymph cells	1.6-2.4	**1.44**	Abnormal

(*) activity unknown

Discussion: Mr. Jeffries' immune tests are significantly abnormal and are abnormal in the same immune distribution. These tests demonstrate an increase in immune dysfunction in all six abnormal areas. The fact that more recent tests demonstrate greater abnormality may be due to a natural fluctuation or it may represent that Mr. Jeffries' immune status is gradually becoming worse. What is important and significant is that this patient's tests are consistently and significantly depressed right across the immune spectrum. The tests are taken in the morning and performed immediately commencing within the hour and tested against a battery of normals. This may also suggest that the patient has an increased risk of falling ill with infectious diseases and possibly malignancy. Since he had no history of prior immune dysfunction before he received the combined Hepatitis A & B immunization, it is not reasonable to assume that this immune abnormality preceded the immunization. However, since he was rarely ill before the immunization, almost no testing was ever done. Also, although the Natural Killer Cell (NK) results for number are normal, the activity is not known. NK activity costs our lab about $800 dollars to perform adequately and then only if we have a battery of several patients to average-out the cost. It is also desirable to repeat the NKC tests to be sure of the results as we have done in the other immune tests. It is impossible to quantify if the killing activity of the NKCells are adequate from this test. He may have a significantly decreased activity or not.

These changes are pathological and are consistent with a subcortical brain injury.

E. 3rd February 2000: Immunosciences Lab: Immune globulin survey:

Immune Test	Patient Result	Normal Reference Range
IgG immune complex	25 ug. Eq/ml ↑	0-20 ug. Eq/ml
IgM immune complex	21 ug. Eq/ml ↑	0-15 ug. Eq/ml
Ig A immune complex	27 ug. Eq/ml ⇑	0-10 ug. Eq/ml
Natural killer cell activity	89.9 LU s ↑	20-50 LU s

(3) *Discussion*: These tests again indicate that there is a significant immune anomaly in this patient's immune status and that probably this patient has a significant immune dysfunction or disease. These tests are non-specific and do not tell us what the immune injury may be. The elevation of Natural Killer Cells is open to debate and may be related to an acute or ongoing infection at the time the blood was drawn. The Immune globulin tests are consistent with the other consistently grossly abnormal immune function tests listed here. When all of the tests noted are taken into consideration, one has to assume that a significant immune

dysfunction exists in this debilitated patient. However, the NKC number tests performed in Ottawa and the NKC activity performed in California suggest that there is an over activity of the NKC as well as an increased number. This could mean: (1) Mr. Jeffries has a malignancy that his body is attempting to destroy; and/or (2) Mr. Jeffries has an acute ongoing or chronic infection that his immune system is attempting to eradicate.

For these reasons if this patient were in Ottawa he should be followed carefully at routine periods, possibly as often as every 6 months in case there is an evolving malignancy or other major illness that can be treated.

F. Immunosciences Lab: 22nd of March 2000

Antiviral and Antiproliferative Pathways

Test	Patient Result	Normal Reference Range in pixels
2-5A Synthetase Activity	3.8 ↑	0.1-1.0
PKR Protein Level by WB	5.3 ↑	0.1 -1.0
Rnase-L Inhibitor WB	8.4 ↑	3.0-5.0
Rnase-L Cleavage assay	16.0 ↑	1.0-10.0

Discussion: These findings are pathological. This series of tests is used by some authorities to determine if the patient has a chronic viral infection and therefore has Chronic Fatigue Syndrome (M.E./CFS). This suggests that it is possible that the enzymes are turned on due to a chronic viral infection. In theory this battery of tests should be activated for any chronic infection and that does not necessarily mean that the patient has CFS but does mean that the patient has a chronic viral infection. It is a presumption by some that CFS is due to a chronic viral infection. All that can be said of this test is that if it is valid then this patient may have a chronic viral infection, or enzymes activated by the Hepatitis A/B immunization or CFS or any combination of the above. What ever the interpretation, it is not a normal finding.

G. RNAse L Protein Determination: 23 Mar 2000: RED Laboratories:

5.7 (normal is less than 0.5 units)

Once again this test is grossly abnormal but is consistent with the tests performed at Immunosciences immediately above. The two sets of tests are consistent and both suggest an ongoing viral infection most likely the result of the decreased effectiveness of his immune system.

H. 8th June, 2000 Children's Hospital of Eastern Ontario; Drs. Byron Hyde & F. Diaz Mitoma

↑ Reticulocytes: 197 (normal range10-150x10 9/L)
↑ Reticulocytes: 213 (normal range10-150x10 9/L) 3 days later

Discussion: The elevated reticulocyte level represents a pathological abnormality. An abnormal increase in reticulocytes is seen when an increase in red blood cell production is occurring as the bone marrow replaces cells lost or prematurely destroyed. It is suggestive of occult disease such as haemolytic aenemia. As noted an increase in reticulocytes can also be seen in a patient who has an abnormal loss of red blood cells. Neither I nor his other physicians have found an indication of frank blood loss in this patient. This suggests a pathological autoimmune destruction of the patient's red blood cells. Reticulocytosis can be seen in leukemia and lymphoma. (N.B. This patient should be routinely followed by his physicians and tested to make sure the above situation is not progressive.)

I. Thyroid Tests: 17 September 1999:
Thyroid peroxidase antibodies: 34.3 U/ml↑ (normal 2.0 U/ml)

Discussion: This suggests that there was a significant auto-immune attack against the patients thyroid gland and conversely serious autoimmune disease. It is this test that led me to believe that the patient had an autoimmune disease such as thyrotoxicosis affecting his thyroid. He also had a distinct 2 cm nodule and abnormal ultrasound of his thyroid and it was for this reason that I suggested a biopsy and further investigations.

J. 16 February, 2000: Immunosciences Lab Results:

Sialoganglioside Antibodies:	25.00 ↑	(normal 0-20 Elisa)
Anti-microsomal Antibodies:	310 ↑	(normal less than 50)
IgG Benzene Ring:	24 ↑	(normal 16) Elisa

Discussion:

Sialoganglioside Antibodies: The positive Sialoganglioside antibodies at + 25 suggests that the patient has either an; (a) unidentified malignancy; (b) a peripheral neuropathy; or (c) a CNS neuropathy. It is a non-specific test and is elevated in a wide number of different malignancies and neurological abnormalities as well as (d) toxic chemical exposures; or (e) all of above. Since we already know from other tests that the patient has an inflammatory CNS anomaly of the CNS from the SPECT study, and also had a malignancy in his thyroid as well as an increasing peripheral paresthesias by history, this test would appear quite an accurate predictor. He should have repeat B12 analysis due to his blood cell pathology and the neurasthesias to make sure he is not also developing a pernicious aenemia (P.A.) or other blood dyscrasia. However, when last tested P.A. did not appear to be a problem although the red blood destruction and the defect in red blood circulation appears pathological.

Anti-Microsomal Antibodies: The anti-microsomal antibody of 310 (normal less than 50) is diagnostic of a thyroid autoimmune pathology such as Hashimotos thyrotoxicosis. When I repeated this test at the Civic Hospital in Ottawa on the 12th of June, 4 months later, the test results were 1:1600 (normal: below 1:80) Even given the possibility of different test sensitivities this is an enormous difference and it would suggest a rapidly evolving autoimmune disease process. One begins to wonder why the earlier thyroid auto-antibodies and positive Sialoganglioside did not make his physicians immediately question the presence of a thyroid

microsomal antibodies: 1:400 (abnormal)

P. September 1st, 2000: Report from Dr. Padma Mangu, Group Health Associates, Cincinnati, Ohio:

During examination I noted a right-sided thyroid nodule measuring 2 cm in size. He was sent to have a fine needle aspiration biopsy, which showed papillary carcinoma of the thyroid. Subsequently, he has since undergone a thyroidectomy.

Q. Iodine-123 Whole Body Scan Imaging: 3rd November 2000: Christ Hospital Cincinnati, Ohio

Report: Two small foci of abnormal uptake are noted in the lower pole region of both thyroid lobes consistent with residual thyroid tissue or previous thyroid mass and less likely adenopathy. The patient is status post previous thyroid surgery for thyroid malignancy. No other foci of abnormal uptake is seen in the chest, abdomen or pelvis.

Discussion: This patient has now had thyroid surgery to remove the cancerous/malignant thyroid containing the papillary carcinoma that was found in his thyroid gland. The significance of this finding is difficult to interpret but may signify that all of the thyroid tissue or malignancy was not removed. He obtained follow up chemotherapy to remove all possible metastatic and localized malignant cells. It is not known if this treatment was successful or will prove successful. *{Also, although I am sure that every precaution was taken to isolate and not remove the four parathyroid glands that are imbedded in the thyroid gland, it might be advisable to recheck his parathyroid activity quarterly over the next year or so in order to ascertain if they are functioning or not.}* Depending upon the sensitivity of the diagnostic imaging small amounts of malignancy or thyroid may have been missed in this test. Only time will tell on the success of these several procedures to eliminate the malignancy.

R. Brain Scans 12 June, 2000 Merivale Medical Imaging: Ottawa

Carotid Doppler: No hemo-dynamic abnormalities of the large blood vessels were noted: Also the Transcranial Doppler of the Circle of Willis and Basilar artery system was normal

Discussion: Dopplers are machines that can actually see the blood vessels and measure the speed of the blood coursing through these vessels. The significance of these two doppler tests is that the Central Nervous System (CNS) problem in this patient does not come from the available blood flow conduits from the heart since all major arteries leading to the CNS are normal and all major blood vessels seen in the brain are also normal. The abnormal brain scans that follow can be concluded to be due to damage either to the microcapillary or small arteries to the brain cells or the brain cells themselves. In some cases we can observe major arterial obstruction to the CNS or in the CNS that are blocked and in some cases major blood vessels outside of the cranium can be repaired. This is not the case with Eric who has a perfusion defect at the level of the basement membrane where blood oxygen and nutrition crosses the barrier to the brain cells. Doppler machines cannot determine pathology at this are but SPECT examination is able to access this level. In conclusion, I found no evidence of large blood vessel injury either in the brain or leading to the brain but did find evidence at a cellular and terminal arterial level and this is discussed now.

1 NeuroSPECT, 9th June 2000, Hotel Dieu Hospital Montreal:

This was the first scan that we performed. It and the second scan were performed on a Picker 300 scan at Montreal. This is the leading SPECT brain-scanning centre in Montreal and possibly in Canada. SPECT scans can demonstrate physiology of brain blood flow at a micro-arterial or capillary level and also brain cell function or dysfunction. SPECT demonstrates changes in anatomical areas but is not essentially anatomical. MRI and CT brain scans demonstrate anatomical structure but except in the newer techniques do not demonstrate function.

This NeuroSPECT demonstrated during the first or circulatory phase, an irregular distribution of the radionuclide mostly in the middle cerebral artery cortex region and involving the temporo-parietal regions. There was a decreased uptake in the posterior fossa structures including the cerebellum and pons. The second or metabolic phase demonstrated similar findings but in addition there was a dissociation between the subcortical and cortical uptake with a lower uptake in both frontal lobes. **This is a pathological scan with elements of vasculitis like pattern.** This will be discussed in a more complete manner after review of the second neuroSPECT.

In my reading of this scan, there appears to be a significant hypoperfusion in the area of the left basal ganglia region. However the area also appears distorted so it may well be on the right side. This finding was not noted by the nuclear medicine specialist reading the scan. (However this abnormality was in both this and the later scan.)

In my reading of this scan there appears to be a more significant change in the middle cerebral artery perfusion on the left side and also in the metabolic phase, suggesting the visual and auditory cortex is more affected than the motor cortex though both sides are affected in a negative manner.

Conclusions: In this first examination significant modifications of brain physiology are demonstrated characterized by an irregular distribution in both middle cerebral artery regions and reduced uptake mostly in the cortical areas of the brain, the temporo-parietal lobe, and the subcortical areas of the brain, the posterior fossa and the pons. There is a vasculitis pattern consistent with an autoimmune reaction affecting the blood vessels of the CNS. As a result, he may also have a similar pathology affecting all of the blood vessels in the body or it may be localized to the CNS.

2 NeuroSPECT, 7th December 2000, Hotel Dieu Hospital Montreal:

The cortical distribution of both middle cerebral arteries is quite irregular. The reduced uptake is most pronounced in the posterior temporo-parietal regions but in both left and right hemispheres. There is also reduced uptake in the posterior fossa, primarily in the cerebellum. The blood supply and perfusion to the pons is normal. This finding remains unchanged and consistent with the scan performed in Montreal in June of 2000.

Discussion: This represents consistent and pathological scans of both the cortex and also subcortical areas of Mr. Jeffries' brain. Mr. Jeffries' brain scan is consistent with an autoimmune encephalopathy. This type of scan is sometimes referred to a vasculitis pattern and can be seen in patients with a vasculitis or an autoimmune reaction involving the cerebral arteries. This is obviously not an acute vasculitis that usually causes death, but it has the same type of pattern and probably represents a chronic form of vascular pathology. We see this type of pattern only in our most chronically ill patients. It is also a typical scan often seen in an HIV, AIDS type

malignancy serious thyroid disease or at least take steps to rule out a thyroid malignancy as a possibility.

Positive IgG Benzene Ring Elisa Test: The result of this positive test of IgG benzene elevation at 24 units (normal 16) is disturbing and highly suggestive of toxic chemical and carcinogenic exposure of benzene (gasoline) that has been incorporated within his cellular structures. Benzene was one of the earliest identified cancer causing chemicals. This benzene was incorporated into Mr. Jeffries body cellular structures and may in part be behind the autoimmune injuries to his thyroid and to his red blood cells and also the inflammatory reaction to his CNS.

The benzene toxic chemical injury is probably only part of his complex illness and disability problem. One should inquire if there is a possible chelating or other mechanism to remove this toxic product from Mr. Jeffries. It should be noted that benzene is lipophilic, accumulating in fatty tissue, notably the CNS.

K. 12th June 2000: Ottawa Hospital; Ottawa Canada:

Microsomal antibodies: 1:1600 (normal: below 1:80)

L. 13th June 2000: Civic Hospital. Ottawa:

Free T4: 10.9 L (normal 11.0-23.0 titres)

Discussion: This borderline figure was one of the rare indications by classical Thyroid tests that were even outside of normal during his early testing in the USA. Often if not always, the T3, T4, TSH was within normal limits when taken here or by other physicians. It was only with the antibody thyroid tests and scans and physical examination and biopsy that we were able to determine the gross malignancy and the Hashimotos Thyrotoxicosis.

M. Iodine-123 Thyroid Uptake Scan Imaging: 13th June, 2000: Christ Hospital Cincinnati, Ohio:

Report: On this report there is a relatively homogeneous uptake of radio-pharmaceutical is noted in the thyroid gland and there is no evidence of hyper-functioning or hypo-functioning nodule.

***Opinion*:** (1) the Iodine-123 thyroid 6 hour uptake value of 15% is in the upper normal range. (2) Mild thyromegaly with early changes of graves disease or goitre. Ultrasound of the thyroid gland may be of additional diagnostic information.

Discussion: This test was inconsistent with the physical examination and prior tests that suggested a Hashimotos Thyroiditis or inflammatory or autoimmune disease of the thyroid. There was no clinical suggestion of Graves disease in this patient. A biopsy was requested.

N. Thyroid Needle Biopsy: 17 July 2000: Drs. Padma Mangu / James Cornwell: Cincinnati Ohio:

Right thyroid: Papillary carcinoma of thyroid. Right side.

O. 17 July 2000: Alliance Laboratories:

encephalopathy. Mr. Jeffries does not have HIV- AIDS, but his brain scan is typical of the degree of pathology that one can sometimes see in HIV encephalopathy. As noted previously, his immune system tests are grossly abnormal. In examination of the actual colour print-outs of the functional brain maps, you can observe the marked irregularities of the outside contours of the brain as well as that of the ventricles. The brain scans themselves demonstrate this lack of symmetry. Not mentioned in this report by Dr. Léveillé is the appearance of a hypoperfusion in the posterior optical cortex in the sagital views (see views 7 & 8 and to a lesser extent in 9 in the 10:32 am views in the circulatory phase). There also appears to be a hypoperfusion in the area of the subcortical areas where the basal ganglia are located. This area again appears to be distorted. The basal ganglia area represents only a small part of this hypoperfused area.

The 12:26 pm views also demonstrate the markedly irregular vasculitis pattern noted in the10:32 am views. These views represent metabolic aspects of the brain cells and once again these views are pathological and represent an encephalopathic condition of his physiological brain architecture. The subcortical architecture is even more irregular and represents a greater degree of pathology than the abnormal cortex.

Neuropsychological Interpretation: In a significantly disabled right- handed M.E./CFS patient we often see a patho-physiological scan involving the left middle cerebral artery. This is part of the brain involving a significant part of the brain's interpretive ability of visual and auditory information. The posterior lobe has multiple functions but one of them is responsible for normal visual interpretation. The information in this occipital lobe is then directed for processing in the area of the left middle cerebral artery circulation area of the brain. However in this patient both left and right sides of the brain appear dysfunctional. The area of the motor cortex, responsible for normal physical movement and response appears pathologically effected. These anomalies alone would suggest that Mr. Jeffries has difficulties in interpretation of visual and auditory information. It is my opinion that these pathological anomalies are totally inconsistent with Mr. Jeffries previous superior ability to perform in every aspect of his high demand pre-illness work life. The clumsiness so obvious when one regards his abnormal balance and walking ability and his wide leg stance is echoed in the abnormal scans of both Mr. Jeffries' abnormal motor cortex and abnormal posterior subcortical brain, in addition to the cerebella patho-physiological features.

One has to wonder about Mr. Jeffries Parkinsonian-like features and stance and walk and the patho-physiological changes noted in the subcortical areas and particularly in the basal ganglia area. This was apparent in both scans of 9th of June 2000 and December 2000. It is unfortunate that there are not better tests to evaluate early Parkinsonian features. At this moment whatever the motion disability problems may be. although they interfere with Mr. Jeffries abilities to walk normally, they cannot be readily placed into the Parkinson category.

In the second scan of December 11th, 2000, there is greater symmetry with equal pathological hypo-perfusion in both left and right cerebral arteries with the motor cortex area hypo-perfusion in this second scan as pathological as in the left cortex.

3. **CNS Vasculitis Sign on Physical Examination:** He has another curious physical finding that should be mentioned. On all visits he has had a twitching movement of his fingers. This is a physical sign associated with CNS vasculitis.

4. PET Scan (Positron Emission Tomography) University of California Irvine Brain Imaging Center:

There are metabolic decreases in the dorsolateral prefrontal cortex. There is metabolic decrease in the right temporal insular cortex. There is a pattern of metabolic hypo-frontality with decreased frontal to occipital rations. **The pattern of abnormalities is compatible with encephalopathy.**

Discussion: Due to the fashion that the software of a PET brain scanner is organized at a primary level is quite easy for a physician, lawyer, judge or member of the general public to read a PET scan, even if they are a novice. There are three coloured printouts. The first page represents a cut relatively high in the brain, the second page a cut in the middle areas of the brain and the third page a cut at a lower anatomical section of the brain.

On each of the pages the upper row of images represents a normal control brain, the second row the patient's brain findings, and the third row the subtraction of the second from the first image. This third row then represents specific areas of Eric's brain that are at variance the normal healthy brain and are thus abnormal or injured. You can readily observe how unusual the patient's brain appears in relationship to the first normal control brain images. In the third row the subtraction is demonstrated. On the fourth row we see the Brodman area numbers so that you can refer back to specific brain areas by number and consult a Brodman guide in order to note the areas of dysfunction. We can observe the following brain anomalies:

On the first page are changes or anomalies in Brodman areas:

View #1 in Brodman areas 4, 6 and 8;
View #2 in Brodman areas 4 and 9
View #3 in Brodman areas 1, 4, 6, 31, 32, 39 and 44.

On the second page there are anomalies in the following Brodman areas:

View #1 in Brodman areas 24 L and R, in 46 L and R,
View #2 in Brodman areas 19, 22, 24 L and R, 31, 46 L and R, and the caudate tail.
View #3 in Brodman areas 22, 37 in putamen, between the caudate and insula,

On the third page there ar anomalies in the following Brodman areas:

View #1 in Brodman areas 10 L & R, 19, 21, 37, hippocampus,
View #2 in Brodman areas 11, 12 L & R, 18, 19, 21, 37 and cerebellum
View #3 in the cerebellum

Discussion: the changes or anomalies mentioned in these several views can be due to a large number of causes and one cannot be precise as to what the cause may be. From a layman's point of view it looks as though Mr. Jeffries' brain had been hit with a shotgun blast of bird shot, the changes or lesions or anomalies are in wide areas of the brain. I have not done an analysis of these changes but the changes in page 3 view 2 and 3 in the cerebellum are quite extensive and reinforce the fact that this patient has a significant movement disorder that is clearly evident in the insurance film of this patient. What is of concern to me is the fact that the changes are so widespread in both the thinking brain and equally in the subcortical or primitive brain that responds to balance. integration and reconstruction of information in the thinking or intellectual brain.

Although I describe these as changes or anomalies they are probably better described as injuries and are most unusual in a young man. The changes are likely due to the autoimmune effects of the Hepatitis B immunization given on top of an existing infection. They could be due to a wide spread vascular injury such as a low-grade infection that could also be due to the Hepatitis B immunization "eternalizing" an existing minor infection; the changes are clearly pathological. Alone with no other proofs, or in someone in their eighties they might be considered expected although unacceptable. However with the history, with the insurance film, with all of the significant numbers of immune and brain changes that I have catalogued in this report, these changes must be considered to be grossly pathological.

5. MRI Brain Scan: Christ Hospital: 29th September 1998:

Report as read by Dr. Thomas McNamara, Professor of Radiological Sciences: UCLA School of Medicine: UCLA Medical Center, Box 951721, Las Angeles California 90095-1721:

Abnormal Scan in that there are non-specific areas of increased signal intensity in the subcortical white matter. These are seen in increased frequency in patients of this age who have the clinical stigmata of chronic fatigue syndrome. It is judged that they are due to CFS filled Virchow Robin spaces. There is a polyp in the left maxillary sinus.

Discussion: This should be repeated with more specific instructions and possibly more modern techniques to outline pathology. Specific areas should be looked at and possibly brain atrophy related to the known problems of cerebella and subcortical injuries. Dr Thomas McNamara read this MRI and noted it to be abnormal due to the subcortical Virchow-Robin white matter changes inferior to the frontal and parietal lobes. He notes that these are seen with increased frequency as an individual ages but in a patient the age of Mr. Jeffries, it is my opinion that these are spaces in the peri-arterial areas that prevent the normal passage of oxygen and nutrients across the cell membrane and into the associated brain areas. In my experience they are seen most frequently in acute onset post infectious type illness.

S. Normal or Negative Tests Results were obtained in the following:

All tests are negative for Toxoplasmosis, Histoplasmosis, Parvovirus B19, and Lyme disease. There were normal serum electrophoresis and protein electrophoresis, rheumatoid and arthritic tests. parathyroid tests.

Summary

Eric Jeffries was totally well and had no disability and no illnesses of which he was aware until he fell ill with what he perceived to be a mild to moderate upper respiratory tract infection two or three weeks prior to receiving a combined Hepatitis A & B immunization on 11th of June 1997.

He received the combined Hepatitis A and B immunizations while still acutely or sub-acutely ill. He then suffered what appears to have been a severe adverse immunization reaction. The pharmaceutical corporation who supplied the immunization termed his reaction a case of serum sickness. It is perhaps better described as a Serum Sickness-Like illness.

Within or about a month after receiving the combined Hepatitis A & B immunization, tests conducted by his physician revealed elevated bilirubin, elevated liver enzymes, an enlarged

tender liver, clay coloured stools and dark urine, all diagnostic of Hepatitis. It is well known that Hepatitis B immunization can provoke a typical hepatitis picture. The fact that the needle biopsy was normal is of no importance since the pathological liver changes could have been focal changes and the area of damage missed. At the same time he had a test result demonstrating cold agglutinins that were indicative of an autoimmune disease. By this same date Eric Jeffries was experiencing increasing cognitive changes, intellectual difficulties and a significant loss of stamina that despite the fact that he continued working, his increasing illness was increasingly affecting his work ability. He still felt this was a short-term illness that his physicians could cure and that he would soon return to full cognitive, intellectual and physical health. Ultimately his illness became such that he could no longer continue in his work. Significantly ill, he was obliged to cease work. He did not get better and then began to search out other physicians to diagnose and treat his illness so that he could return to work.

Mr. Jeffries illness has been described as a CFS type illness. Essentially M.E./CFS type illnesses tend to injure the software and communication capabilities of the CNS. The M.E./ CFS illnesses act like any disease with a wide range of penetration and varied clinical aspects. It is my experience that patients who fall ill with a M.E./ CFS like illness at the same time as an immunization injury tend to be more disabled than either a routine CFS or routine immunization injury. Whether Mr. Jeffries is best described as CFS or Post-immunization illness or other autoimmune CNS injury is really simply a matter of terminology. I have demonstrated by the tests we have conducted that Mr. Jeffries has immunological and physiological injuries consistent with his complaints and consistent with the disability that he describes. The fact that I was able to diagnose in greater detail than many of the physicians who Mr. Jeffries consulted is simply a factor of the time and scientific technology I have used and the excellent physicians and researchers that I have been able to call upon to assist me in this work.

There is another problem that vexes me. Eric Jeffries has a father with two genetically inherited CNS illnesses. Usher Syndrome and Retinitis Pigmentosa; each of these progressive illnesses have a wide range of expression and may have left Eric more vulnerable to a form of CNS disease. These illnesses are in part related to loss of intellectual ability and to ataxia or balance problems. Eric is experiencing both problems. But it is too early to know if Eric's multifaceted illness is in any way related to the above two debilitating hereditary illnesses. Eric has one full sibling who has a significant autoimmune disease of lupus or a lupus like disease with a positive ANA. This genetic pathology puts Eric in a potential increased risk of developing an autoimmune disease process and Eric Jeffries certainly has a significant autoimmune problem as we have demonstrated. This pathological connection remains to be investigated and should be better explored. I should note that at the moment medical science has no means of treating either Usher's Syndrome or Retinitis Pigmentosa, both are severely disabling illnesses.

Eric suffered from Rocky Mountain Spotted Fever (RMSF) when he was a 4 year old child. The predominant injury with RMSF is a perivascular injury in almost any organ. I am not aware whether this vasculitis compromises local perfusion, in which significant vascular injury can occur. The milder clinical symptoms and pathology of RMSF are similar to those that Eric Jeffries suffers from today. The mild RMSF symptoms are the following:

> severe headache, malaise and myalgia, chills and rigors, stiff neck, nausea, abdominal pain and tenderness, lethargy, conjunctival suffusion, photophobia, cough, confusion, liver changes, a petechial rash usually on the extremities but also on the trunk, similar to that seen in the photograph of Eric's axillary area. Brain injuries include focal vasculitis or perivascular infiltrates.

It is my belief that these problems noted above would give a SPECT scan potentially similar or identical to those found in Eric's scans. The long-term sequellae of RMSF usually occur a year or so after the illness and include paraparesis, peripheral neuropathy, bladder and bowel problems, cerebellar, vestibular and motor dysfunction, language disorders and scrotal pain. Eric also has an ongoing autoimmune injury to his red blood cells. I can find no mention of red blood cell injury associated with RMSF, only white blood cell and platelet injury.

I outline this delayed RMSF profile, because these symptoms are identical or close to the Eric's complaints in the descriptions of his illness. It is quite possible that the original injury with RMSF has left an antibody receptor marker on these affected areas, and specifically his small arteries and these markers have somehow been stimulated by the infection and the combined Hepatitis A & B immunization. The SPECT brain scans demonstrate a vasculitis like pattern in his brain as would be expected in a mild to moderate RMSF.

Eric describes a series of symptoms, particularly his Neuropsychiatric and muscular and neurological symptoms that are incompatible with maintaining his work at any level. These symptoms are consistent with the laboratory and scientific tests conducted on Eric by myself and his other physicians.

Eric has a falling pulse pressure from 40 cm of mercury to 15 cm of mercury that is consistent with dysautanomia. Effectively, what this means is that as long as he is lying down he has enough blood pressure to supply his brain, muscles and gut adequately with circulating blood. As soon as Eric stands there is a decrease in blood pressure such that he may have insufficient blood to keep his brain supplied with adequate oxygen levels. The fact that Eric is over 6 feet tall makes this haemodynamic problem even more exaggerated. These pathological changes were also apparent when I had his levels of circulating blood volume tested.

Eric appears to have ataxia. lethargy, a shuffling walk, and body habitus consistent with a Parkinsonian-like illness or a CNS injury affecting the cerebellum, basal ganglia, or posterior columns of the spinal cord. I have demonstrated exactly these types of changes or injuries to these areas of his CNS in the SPECT scans.

Eric has a neurological examination and speech changes consistent with a CNS dysfunction and with an autoimmune illness or other illness affecting his CNS. He has a CNS or motor illness consistent with a non-Parkinsonian movement disorder.

Eric has an elevated reticulocyte count and has no obvious blood loss and this is consistent with an autoimmune injury destroying his red blood cells. The early elevated bilirubin in the first month of his illness in the absence of reticulocytes is consistent with a hepatitis but the later elevated bilirubin with normal liver enzymes and elevated reticulocytes is also consistent with excessive red blood cell destruction.

The investigation of Eric's thyroid pathology revealed an overwhelming autoimmune disease attacking Eric's thyroid gland. Physical examination and further testing revealed the presence of Hashimotos Thyroiditis that is itself an extreme autoimmune disease or injury affecting at least part of the endocrine system. Further investigation revealed a potentially fatal malignancy of the thyroid. His thyroid was removed and the patient placed on radiation and then, hormone replacement therapy. It is not known if all of the malignancy was removed successfully. Only time will tell. It is not known if the four parathyroid glands are still functioning. It was believed that up to two of the four glands were removed. It is not known if the blood supply to the remaining two was compromised or not. Often it takes time to balance the thyroid replacement

effectively and in a few, patient's hormone treatment may never be effective. It is technically possible that the autoimmune response may diminish the effect of the replacement hormone.

The only other glands that are readily visible to examination are his testes and these appear unusually tender and thus possibly inflamed. I cannot rule out an autoimmune inflammatory effect on the testes and/or adrenals or other glands.

We already know that his effective blood circulating pressure is probably insufficient when Eric is standing. This may decrease the circulating thyroid replacement to his end organ cells. We will shortly discuss the grossly abnormal circulating red blood cell and plasma volumes that will make further exaggerate this problem of adequate circulation.

I reviewed the grossly abnormal circulating red blood cell and plasma volumes. The red blood cell volumes available to carry oxygen to Eric's brain and muscles and gut are in the range of 85% when he is lying down, which is sufficient while sleeping but this would give a figure of as low as 75% when sitting and possibly as low as 65 % when standing. These levels would be insufficient to supply the brain with oxygen when Eric is physically or mentally active. To date we have found no way to correct this anomaly.

This problem is compounded by the dramatically falling pulse-pressure noted in this report.

When the abnormal circulating blood volume and abnormal falling pulse pressure abnormality is compounded when he is standing, the oxygen deprivation to his CNS may be even more exaggerated. It should also be remembered that the blood flow works on a series of presso-receptors that further regulate the blood flow. The heart is protected at all times and when the blood pressure falls and when the circulating red blood cell volume falls the body survival defences trigger a reaction that deprives the brain, muscles, organs and gut of blood to make sure that an adequate blood supply and oxygen is maintained to the heart muscle. This is obviously happening to Eric and this simply deprives Eric's brain of the necessary oxygen source to carry on. When this happens, not only does the brain not have sufficient oxygen to operate effectively, but the normal cell breakdown products not only increase due to oxygen deficiency but these cell break- down products cannot be removed at a reasonable rate. To prevent destruction of the brain cells, the brain acts by simply shutting down. If the brain did not shut down the cells and the brain would be permanently injured.

Eric's clinical history is strongly suggestive of such a shut down.

Eric's measured circulating plasma volumes are even worse than his circulating red blood cell volume deficiencies. Even when Eric is lying down he is running at 72 % of the normal circulating plasma. To name only two of a plethora of essential brain chemicals, the plasma carries insulin and growth hormone that is essential for normal cell maintenance and energy. When Eric stands the available plasma circulating volume falls to a figure of 52-62% of normal. This would also cause the available thyroid hormone to potentially reach the end organ cells at a rate of only 50% of the required maintenance dosage to maintain normal metabolism. What this means of course is that a normal amount of thyroid replacement hormone for Eric's weight may only be 50% of what his body requires. In other words his thyroid hormone replacement may require a significant upward adjustment to supply the essential body metabolism requirements. This equation is probably true of all of his essential body chemicals and may be another factor why Eric has been essentially turned into a zombie, a slowly walking shell of his former self. Eric is severely disabled.

He appears to have an autoimmune illness affecting his CNS vascular system as well as his red blood cells and it is reasonable to consider that the coronary arteries and microcirculation of his heart if not already affected will be affected negatively in the future.

The Natural Killer Cells level and function should be followed closely on a quarterly basis since we do not know if all of the thyroid malignancy was removed, although all the thyroid was removed. I am a little troubled by the hot spot that appears to still be in the area of the thyroid after the gland was removed. Also, we do not know if there are other malignancies since it is quite possible that the thyroid malignancy was the result of an immune response to the immunization during a time when the patient was obviously carrying an unknown infectious disease and that may have been rendered invisible by the capsular antigen sequence incorporated in the Hepatitis B component of the immunization he received. Unfortunately the Hepatitis A component is relatively new on the market and I simply do not know of the negative effects of this immunization if there are any. Hepatitis A is a picornavirus, anatomically identical to polio and coxsackie and ECHO virus but with a slightly different internal gene sequence. These enterovirus are all potentially neurogenic and hepatic viruses. The trials on humans of Hepatitis B immunization only followed the immunized patients for the first 72 hours. This is well below the time necessary to establish short or long-term injury. This is well below the time when Eric developed his complex disease processes or at least when these changes were observed. I do not know the time of the trial the Hepatitis A immunization was followed but it is likely that it was the same as for Hepatitis B and this was insufficient.

The tests on antiviral pathways are consistent in both the Immunoscience Laboratory tests performed in California and also those performed in the RED lab in Belgium are consistent with the fact that Eric has a chronic viral infection or antiviral pathway triggered by the Hepatitis A/B immunization. This is also consistent with the abnormally elevated NKCells number and activity noted earlier. It is my opinion that Eric has a chronic viral infection of unknown nature and until he rids himself of this probable virus or virus types or antiviral triggering mechanism he will remain ill and disabled as well as potentially progressively ill and progressively subject to further autoimmune insults. Anti-viral therapy is non-specific and I am not aware how to treat such a problem effectively.

I also note the Sialoganglioside abnormality. This test finding is suggestive of ongoing neurological injury or possible malignancy. I also note that although the B12 levels were normal I do not know if Eric was taken exogenous B12 and whether this is a false positive. It may well be normal but it too should be followed due to the apparent red blood cell destruction process that appears to be active.

The abnormal IgG benzene test is equally troubling since it may have been a potentiator to the immunization associated with immune damage or immune instability. It is too early to effectively repeat these tests but they should be done quarterly to ascertain in what direction they are going.

The two NeuroSPECT examinations performed in one of Canada's leading brain imaging centres are both grossly abnormal and represent an abnormal blood perfusion to the patients brain cortex, that is responsible for normal intellectual sensory and some motor processes. The subcortical changes are also pathological and represent physiological injuries to both the recruitment areas of the brain, hormonal control centres and balance and primitive centres. This can better be evaluated by PET scans using various modalities but this is also very expensive and although it adds to our diagnostic understanding it does not help the patient get better. When the

SPECT scans were repeated and were essentially identical and so we can assume were accurate and although they were taken approximately 6 months apart, demonstrate no physiological improvement in brain function what-so-ever.

Neuropsychological Assessment: Once again this was grossly abnormal and incompatible with any work in and intellectual, competitive and energy-demanding field of employment.

These are my conclusions to date if other tests or information come to my attention I will supplement this report and these opinions. It is possible that individual tests or clinical findings and consequent opinions may be questioned, however, the incredible number of historical, clinical and test results all are consistent with a major and overwhelming injury to the immune and CNS systems. There is also significant evidence of immune and endocrinological injury that has been discussed. I doubt if we have found all of the injuries that have occurred to Eric and that have caused his ongoing and chronic injuries and disability. Unfortunately, I have no suggestions for any restorative treatment or treatments to return Eric to his former excellent health. To my knowledge, there is no accepted North American treatment for his health problems. I will continue to search for one.

The relationship of the CNS injury and the Hepatitis A and B immunization is based upon my own personal observations concerning over 200 patients who have experienced adverse reactions to Hepatitis B immunization. I have seen only one other patient with an adverse reaction to Hepatitis A immunization. I have therefore no significant experience with adverse reactions to Hepatitis A and significant experience of adverse reaction to Hepatitis B. The manner of Eric Jeffries falling ill is fairly typical of a large number of the adverse reactions I have seen after Hepatitis B immunization as are the CNS changes. The immune abnormalities are also consistent with this type of injury. The abnormal thyroid autoimmune disease is also fairly typical, however I have never seen a case of thyroid malignancy following Hepatitis B immunization.

It is my opinion that Eric Jeffries is totally disabled intellectually and physically and has been so disabled since he ceased work over three years ago. He has demonstrated no signs of improvement during the past three years or more. For me the question is whether his complex illness will be progressive or if it has or will plateau. I am not aware of any treatment for his complex illness. Early in his illness he might have been assisted by a trial of corticosteroids but when later in his illness this was attempted it was of no benefit. I am not aware yet of the role that the genetic diseases described have in his overall pathology and disability. His is a most unfortunate case. It is my opinion that Eric Jeffries should not be subjected to any immunization other than tetanus immunization and particularly he should avoid for the rest of his life both Hepatitis A and B immunizations. It is my opinion that this Hepatitis A and B immunization triggered his several illnesses and is part of his ongoing and complex disabling illnesses. Eric Jeffries has an obvious motor disturbance and intellectual disturbance and these are part of his general illness. They have been discussed in detail.

Yours Sincerely,

Byron Hyde M.D

Byron Hyde, M.D.